not R wild oat leaves. Incorporation of [^{14}C]malonic acid showed that de-novo synthesis of short-chain fatty acids was not affected by tri-allate or tri-allate sulfoxide in R or S plants. In contrast, tri-allate sulfoxide was equally inhibitory to fatty acid biosynthesis and elongation in R and S plants, indicating that elongases in R plants were still sensitive to the activated tri-allate sulfoxide. The results further support the hypothesis that reduced tri-allate sulfoxidation in R plants is the primary mechanism of resistance.

To determine the inheritance patterns of resistance, we made crosses between the inbred R and S lines described above.[10] Seeds from R × S reciprocal crosses and F_2 populations were treated with 0·275, 0·55, and 1·1 kg ha^{-1} tri-allate in the greenhouse, and plant heights recorded after 37 days. Heights of R and S parental plants averaged over treatment rates were 62·5 cm and 0.2 cm, respectively, providing unambiguous classification of phenotypes. Plant heights of F_2 progeny were bimodally distributed with 5·6% and 94·4% of the plants classified as R and S, respectively. Chi-squared tests showed that the observed segregation ratios were consistent with an inheritance model that tri-allate resistance is conferred by two recessive nuclear genes.[10] Inheritance ratios were not affected by treatment rate or parental source of resistance.

Because we have been unable to assay a tri-allate sulfoxidase activity, we do not know if reduced rates of sulfoxidation in R plants are due to lower enzyme levels or to an altered substrate specificity. However, since resistance appears to be controlled by two recessive nuclear genes, we propose that two enzymes are required for tri-allate sulfoxidation, and that mutations in both genes are required for resistance. This report marks the first documented case of herbicide resistance in plants conferred by a reduced rate of metabolism. Further, we know of no other examples of resistance to insecticides, fungicides or antibiotics that are due to a similar lack of metabolic activation. Characterization of tri-allate-resistant wild oats thus represents a novel mechanism of pesticide resistance, and illustrates the diverse strategies by which organisms are able to escape or overcome strong selection pressures in the environment.

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Insecticide Resistance Management in Europe: Recent Developments and Prospects

Ian Denholm[1] & Jørgen B. Jespersen[2]

[1] IARC-Rothamsted, Harpenden, Hertfordshire AL5 2JQ, UK
[2] Danish Pest Infestation Laboratory, Skovbrynet 14, DK-2800 Lyngby, Denmark

Background

Problems with insecticide resistance in Europe have increased markedly in recent years as a consequence of both the accumulation of resistance mechanisms by some species and the greater diversity of pests attacking valued commodities, especially in the horticultural sector. Arthropods of particular concern include lepidopteran, aphid, whitefly and mite pests of agriculture and horticulture, coleopteran and mite pests of stored grain, and public health and livestock pests including mosquitoes, houseflies and cockroaches. Highly polyphagous thrips and leaf-miners constitute potentially severe problems that are less clearly resolved in a European context. It is also becoming apparent that increased trade in plant material between European countries, and between Europe and overseas, is not only expanding the geographical range of many pests but is also accelerating the spread of resistance genes.[1] The complexity and implications of resistance problems are illustrated well by recent work on two particularly important agricultural pests—the peach-potato aphid, *Myzus persicae* Sulzer, and the cotton or tobacco whitefly, *Bemisia tabaci* Gennadius.

Resistance in Myzus persicae

M. persicae causes direct feeding damage on many crops, but, more importantly, is a major vector of

several virus diseases. As a result, it has been subjected to extensive insecticide treatment, and populations throughout the world have developed resistance to organophosphate (OP), carbamate and pyrethroid insecticides. Until recently only one mechanism had been identified: the overproduction of one of two closely related carboxylesterases, E4 and FE4, that degrade and sequester insecticidal esters.[2] This is now known to reflect a progressive amplification of structural genes encoding these enzymes.[3] Bioassays and field trials indicated that, although this mechanism conferred resistance to almost all available aphicides, resistance was least expressed to carbamates, including pirimicarb.[4] Pirimicarb consequently assumed a key role in control strategies in many European countries since it offers the best prospect of contending with high levels of carboxylesterase-based resistance late in a cropping season.

In 1990, a new resistance mechanism based on an insecticide-insensitive form of acetylcholinesterase (AChE), the target site of OPs and carbamates, was identified in *M. persicae*.[5] This confers strong resistance very specifically to pirimicarb and to triazamate, a novel triazole aphicide. It was first detected in an FE4-overproducing clone originating in Greece in 1990. Since then, work at IARC-Rothamsted has also documented its occurrence in Japan and a northward expansion in its European distribution.[6] In 1996, many strains collected at sites in eastern England reporting control difficulties with pirimicarb were confirmed to possess this insensitive AChE variant. This discovery has a profound bearing on the management of resistance in *M. persicae*. Recommendations to exploit pirimicarb to combat E4 and FE4 resistance now risk accelerating the selection and spread of the new mechanism, thereby rendering this chemical ineffective over large areas. Ironically, this would also have severe repercussions for the efficacy of triazamate—one of very few novel aphicides available against *M. persicae* in Europe. Future attempts to combat resistance in this species must clearly place greater emphasis on more careful use of pirimicarb, based on detailed monitoring to document the dynamics and geographical spread of the new gene. One major research objective has therefore been to develop biochemical and DNA assays for diagnosing all resistance mechanisms known to occur in this species.[6]

Resistance in Bemisia tabaci

Over the last ten years *B. tabaci* has undergone a dramatic expansion in its geographical range and pest status. In addition to increasing in importance in the tropics and subtropics, it has spread into temperate countries, including those in northern Europe. Infestations in the latter (including the UK) involve a novel biotype (the so-called 'B-type') and are associated especially with the expanding international trade in ornamental plants.[7] Once introduced into protected environments, it has the potential to disrupt established IPM practices and spread onto a wide range of crops vulnerable to the viruses and physiological disorders transmitted by this species. The proven capacity of *B. tabaci* to develop insecticide resistance poses another potential threat to the containment of this species in Europe.

Strains from throughout the world have been tested with insecticides representing the major insecticide groups to assess the extent, breadth and consistency of cross-resistance patterns. Results demonstrated the widespread, almost ubiquitous occurrence of strong resistance to OPs and pyrethroids.[8] It is notable that strains intercepted sporadically on plant material reaching the UK exhibited comparable patterns of resistance to ones from the Netherlands, southern Europe and the Middle East—all possible sources of UK infestations.

Potential implications of transferring insects between countries are best exemplified by work with the insect growth regulator buprofezin. This compound was first released in the Netherlands in the late 1980s, and rapidly gained favour against glasshouse populations of whiteflies resistant to most conventional insecticides. By 1991 there were already reports of reduced control with buprofezin, which were subsequently attributed to buprofezin resistance.[9] Further testing of Dutch strains showed resistance to be well established in the Netherlands, a consequence of relying excessively on a single product under environmental conditions promoting rapid population growth and build-up of resistance genes. More surprisingly, resistance to buprofezin was also detected at varying frequencies in strains from UK glasshouses, prior to the official approval of buprofezin in the UK. The most likely explanation is that genes conferring buprofezin resistance had been imported into the UK from countries where this chemical was already in use.

Formation of ENMARIA

These and other case-histories emphasise the importance of international collaboration to standardise technology, exchange data, minimise duplication of effort, and co-ordinate resistance management recommendations for Europe as a whole. Initiatives under way to harmonise approaches for assessing resistance risks and extending management guidelines within a unified EU regulatory framework[10] reinforce the need for a Europe-wide perspective on resistance monitoring and management. This being so, we are pleased to announce the launch of a European Union-funded Concerted Action involving 13 countries and entitled 'ENMARIA: European Network for the Management of Arthropod Resistance to Insecticides and Acaricides'. The agrochemical industry, through its Insecticide Resistance Action

Committee[11] is also participating actively in this initiative. The first ENMARIA workshop took place in conjunction with Resistance '97 at Rothamsted in April 1997, when plans for sharing data and experiences, compiling a European resistance database, and organising further meetings and training visits over the next 30 months were discussed and implemented. ENMARIA will facilitate the dissemination of results of nationally funded programmes as widely and rapidly as possible across Europe. Involvement in ENMARIA is open to all interested individuals and organisations; further details are available from the main co-ordinators whose details are listed below:

Dr Jorgen B. Jespersen
Danish Pest Infestation Laboratory
Skovbrynet 14
DK-2800 Lyngby
Denmark
Tel: (45) 45 878055
Fax: (45) 45 931155
Email: j.b.jespersen@ssl.dk

Dr Ian Denholm
IARC-Rothamsted
Harpenden
Herts. AL5 2JQ
United Kingdom
Tel: (44) 1582 763133
Fax: (44) 1582 762595
Email: ian.denholm@bbsrc.ac.uk

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Development and Implementation of Biochemical Insecticide Resistance Detection in Danish Field Strains of *Musca domestica*

Michael Kristensen, Andrew Spencer & Jørgen B. Jespersen

Danish Pest Infestation Laboratory, Ministry of Food, Agriculture and Fisheries, Lyngby, Denmark

This summary discusses a project, the overall objectives of which are to identify the main resistance mechanisms, to develop and implement biochemical methods for studying them and to implement resistance management strategies for the most important agricultural pests in Denmark. In the initial phase of the project we have focused on insecticide resistance in houseflies caused by metabolism by general or specific esterases, by glutathione-*S*-transferase (GST), or by P450 monooxygenases, and by alteration of the organophosphate target acetylcholinesterase (AChE).

Monitoring of resistance in Danish field populations of houseflies (*Musca domestica* L.) related to the use of insecticides for fly control has been performed yearly since 1948.[1] Field-collected strains with different resistance patterns have, during this time, been isolated and are regularly selected. In this summary, we compare three *M. domestica* laboratory strains, the susceptible WHO, the multi-resistant 381zb and the azamethiphos-resistant 594vb, which were tested using biological and biochemical assays. We measured the enzymatic activity towards four naphthyl ester and three *p*-nitrophenyl ester substrates, GST activities with 1-chloro-2,4-dinitrobenzene (CDNB) and 3,4-dichloronitrobenzene (DCNB), monooxgenase activity with *p*-nitroanisole (PNA) and inhibition of AChE by azamethiphos and methomyl.

With dimethoate the 381zb strain has an R/S ratio of 59 at LD_{50} and 56 at LD_{95}, which is synergised by neither DNF nor PBO. AChE activity of 381zb is less sensitive to inhibition by azamethiphos and methomyl